

CHILDHOOD RESPIRATORY DISEASE

Perhaps no claim regarding ETS is as capable of provoking strong feelings as the charge that parents who smoke may harm the health of their children. While the issue of parental smoking is laden with emotion, the scientific basis for the claim is difficult to interpret. For example, while one study examines respiratory symptoms or illness such as coughs and colds by questionnaire responses from parents¹, another measures lung function with special equipment at a school or health facility.² In the U.S. alone, according to one report, this has led to a situation in which studies of ETS and the respiratory system are "being carried out by at least three different groups, are employing different populations and methodologies and have led to varying conclusions."³

Perhaps not surprisingly, such studies, each with a different sample size, data collection method, and analysis, tend to yield factually incompatible and contrary conclusions. For instance, although certain studies have reported adverse findings between parental smoking and respiratory illness in children,⁴⁻⁴⁷ or invasive bacterial or viral infections⁴⁸⁻⁴⁹ others have observed no significant relationship.^{1,50-65} After a five-year study of over 400 children, for example, a Dutch research group concluded there was "no evidence" that parental smoking had an appreciable effect on respiratory symptoms in school children.⁵⁷ A similar conclusion was

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reached by a group of U.S. researchers, including a critic of smoking, who found "no significant relation" between parental smoking and respiratory symptoms in a study of nearly 400 families with 816 children in three cities.⁵⁰

The contradictory nature of findings on the issue of parental smoking is also apparent in the growing number of studies examining the relationship between parental smoking and children's respiratory or lung function. Although several reports have claimed that parental smoking results in decreased pulmonary function in children,^{3,13-14,32,67-81} others have not,^{82-88,66} including those of a U.S. research group who have published a series of studies on this subject.^{2,89-91} In 1982, for example, the U.S. group showed that a comparison of body size with lung function eliminated any reported correlation between parental smoking and children's lung function.² Two years later, a reanalysis of data from families in their study population again showed that "parental smoking did not have a significant effect on children's pulmonary function; smoking habits of others in the household (predominately siblings) did not have any effect either."⁹⁰

Moreover, the authors of studies reporting adverse effects from ETS exposures among children concede that their conclusions must be viewed with caution because of numerous

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confounding factors. The potential impact of such factors was given special consideration in the report from a workshop on ETS sponsored by the U.S. National Institutes of Health.³ After listing numerous such factors, including types of heating used, socio-economic status and demographic and medical characteristics of the study population, the report cautioned "that any study which ignores them will be seriously flawed."

The importance of such factors in evaluating the outcome of research on parental smoking is supported by a number of reports which have shown that the use of gas stoves in the home may be independently associated with respiratory disease^{40,54,89,92-96} and impaired pulmonary performance^{97,98} in children. One group of British researchers acknowledged the possible influence of factors such as cross-infection in the home and genetic susceptibility to childhood respiratory illness and symptoms.¹¹⁻¹² More recently, researchers in Hong Kong reported "a highly significant correlation" between the frequency of respiratory illnesses of mothers and their children.⁹⁹ One study reported that children with recurrent respiratory tract infections "tend to belong to families with health problems."¹⁰⁰ Maternal age has also now been recognized as a possible factor in childhood lower respiratory illness.¹⁰¹

Other confounding factors independent of parental smoking have been reported recently in the literature. For example, studies in the United Kingdom have identified damp housing^{60,102-105} and paternal occupation¹⁰⁶ as potential explanatory factors for the occurrence of respiratory illness in children. Other recent studies have identified outdoor air pollution,¹⁰⁷⁻¹¹⁰ infections transmitted during day-care attendance^{63,111-114} decreased breast-feeding,^{113,115} and the use of kerosene heaters and woodburning stoves in the home and exposure to wood smoke outdoors¹¹⁶⁻¹²⁰ as factors related to childhood respiratory disease.

The relevance of dampness in the etiology of respiratory symptoms in children is supported by current research which links dampness with the presence of molds, dust mites, fungi and other allergenic microbes. The growth of fungi and molds in the home is directly related to respiratory symptoms and sensitization reactions in some individuals.^{104,121-124} Recent investigations report, moreover, that exposure to ETS does not increase sensitization to common allergens in children.¹²⁵⁻¹²⁶ Exposure to indoor sources of NO₂ has also been associated with respiratory symptoms and decreased pulmonary function in children.¹²⁷⁻¹²⁸

Others have conceded that the reliance of such studies on questionnaires for information about respiratory symptoms casts

doubt on their reported findings. In one study that reported a significant association between parental smoking and respiratory symptoms, for example, it was noted that even "slight changes" in the way the questions were phrased could result "in substantial differences in the type of responses one obtains."¹⁴ Similarly, another study observed that there was a significant difference in the respiratory symptoms reported depending on which parent completed the questionnaire.²⁴ Authors of another study that reported adverse effects of parental smoking on the respiratory health of children conceded that "since the exposure variables used in these analyses were subject to substantial measurement error, a more refined measurement of personal exposure is required."²⁹ One researcher who is critical of parental smoking has stated that "quantitative assessment of involuntary exposure of infants and children to ETS has been very imprecise and probably inaccurate."¹²⁹

Studies utilizing seemingly more objective standards such as actual measurements of lung function are also open to criticism. Even reviewers of the literature who are critical of parental smoking concede that the tests used in these studies are "influenced by a large number of variables."¹³⁰ They list age, height, and gender of the test subject as well as his or her motivation, cooperation, and effort put forth during the test, the skill and experience of the operator, and the type of

instrumentation used as variables that can affect the results of pulmonary function measurements. The reviewers explain that these problems are especially important in pulmonary function measures taken in children. In 1989, two American co-researchers, Witorsch and Witorsch, reported that "it has been shown that the mean pulmonary performance within a single group of children can vary significantly from one spirometry test to the next without any apparent cause" and that it "is noteworthy that such statistically significant differences are similar in magnitude to most of the small decrements in pulmonary function reported in children of smoking parents."¹³¹

The shortcomings of studies analyzing the relationship between ETS exposure and childhood health were highlighted in a 1988 report by two U.S. investigators who re-examined 30 such studies and evaluated them for their scientific validity.¹³² They noted that while several studies of adequate scientific design had reported a statistically significant relationship between ETS exposures and childhood health, "most studies had significant design problems that prevent reliance on their conclusions." The authors concluded that "many questions remain, and future studies should consider important methodological standards to determine more accurately the effect of passive smoking on child health."

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Thus, claims that parental smoking plays a causal role in the development of respiratory symptoms and reduced lung function in children are not scientifically justified. Such claims are typically based upon a single study of a selected symptom (such as cough or wheeze). These kinds of studies invariably fail to consider nutrition, health habits of the family, and other lifestyle variables. Similarly, studies that report reduced lung function in children of smoking parents fail to address the issue of socio-economic status or the potential role of genetic and family traits in pulmonary function capabilities.²⁵ Moreover, the reductions reported in the literature are small and of uncertain clinical or biological significance, and are contradicted by a number of studies that reportedly have observed no effect of parental smoking on children's lung function.

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The majority of the epidemiological studies of parental smoking and childhood asthma utilize questionnaires to estimate a child's exposure to ETS. However, there are several studies (most of which are fairly recent) that measure cotinine levels in bodily fluids to estimate a child's exposure.^{22,25,31} These studies report a statistically significant association between parental smoking and asthma in children. However, there are several problems with the use of cotinine levels to estimate ETS exposure.

While some reports may suggest that cotinine is a reliable marker for total exposure to ETS, many others do not for a variety of reasons.⁴⁰⁻⁴⁹ For example, it has been reported that individuals metabolize nicotine in different ways at different times and that elimination rates for cotinine vary among individuals. In addition, recent research indicates that diet may contribute to levels of nicotine and cotinine found in the body, thereby interfering with reported exposure levels from nicotine in ambient air.⁵⁰ Scientists have also noted that different methods of analysis may influence final recorded levels of cotinine.⁵¹ Finally, it has been observed that because nicotine is largely present in the gas-phase of ETS, measurement levels of its metabolite, cotinine, do not reflect exposures to other constituents present, for example, in the particulate phase of ETS.⁴²

There is at least one group of researchers that has performed clinical studies of the possible association between exposure to ETS and exacerbation of asthma in children. In 1990, German researchers Oldigs et al., reported that exposing children with bronchial asthma to cigarette smoke sufficient to reach a level of 20 parts per million (ppm) carbon monoxide for one hour did not affect their lung function or bronchial responsiveness.³⁶ These authors have reported similar data in subsequent studies.³⁸

Other authors, utilizing questionnaires to estimate exposure, have reported similar findings. In 1990, Charles Sherman and his co-investigators reported that neither paternal nor maternal smoking "bore an apparent relation to the development of asthma" in a sample of children, aged 5 to 9, enrolled in public and parochial schools in East Boston, Massachusetts in 1974.³⁷

In 1992, Martinez et al. reported that childhood asthma was associated with maternal smoking in children whose mothers had 12 or fewer years of education.⁵² However, they reported that there was no association between maternal smoking and asthma in children of mothers with more than 12 years of education. While not suggested by the authors of this study, these data could be interpreted as providing support for the theory that socioeconomic differences are important confounders in studies of parental smoking and childhood asthma.

A British researcher, Strachan, has implicated damp housing as a potential factor in the development of childhood asthma.⁵³ After controlling for the possible influence of housing tenure, number of people per room, number of smokers in the household, and gas cooking, he reported that the relationship between damp housing and childhood asthma in his study population remained "highly significant."

Another group of authors, Horwood et al., reported that parental smoking habits were not significantly associated with the development of asthma in a birth cohort of New Zealand children.³⁴ The authors concluded that "asthma in early childhood appeared to be inherited to some extent, its age of expression was related to the child's sex, and it had a complex interaction with other forms of allergic disease."

In another study of New Zealand children, Mitchell et al. reported that the following factors appeared to be precipitating factors for asthma attacks: 1) weather (70%); 2) infection (61%); 3) stress or excitement (25%); 4) dust (24%); 5) pollen (17%); 6) food (13%); 7) running out of medicines (11%); 8) animals (10%); and 9) exercise (4%).⁵⁴ A "miscellaneous agents" category, including passive smoking, noncompliance, etc., was reported to

be associated with the precipitation of asthma attacks in only four percent of patients.

In summary, there are many potential confounding variables which should be controlled for in any study which purports to show a relationship between parental smoking and childhood asthma. In an article which argues against parental smoking, the author concedes, "the relative risk or odds ratio in the larger studies which controlled to some degree for confounding has been modest, of the order of 1.5" and that confounding "is a consideration wherever the measure of effect is modest."⁵⁵

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